on Cell Outgrowth in Culture of Normal Rat Fibroblasts and Rat Benzpyrene-Induced Sarcoma, Cancer Research, 2:28-31, 1942.
12. Hektoen, L., Further Studies on the Effects of Roentgen Rays on Antibody Production, J. Infect. Dis., 22:28-33, 1918.
13. Lusztig, A., Die Wirkung der Röntgenstrahlen auf die Spezifischen Immunkörper, Zentralbl. Bakt. Paras. und Inf., 1 Abt., orig. 111:244-266, 1929, cited in (5).
14. Lasnitzki, L., Effect of X-Rays on Cells Cultivated in Vitro, Brit. J. Radiol., 16:61-67, 1943.
15. Packard, C. E., The Biological Effects of Short Radiations, Quart. Rev. Biol., 6:253-280, 1931.
16. Patterson, E., Comparison of the Action of X and Gamma Radiation on Fibroblasts, Brit. J. Radiol., 15:257-263, 1942.
17. Rahn, O., in Glasser, O. (editor), Medical Physics, Year Book Publishers, 1944.
18. Rosselet, A., and Humbert, R., Roentgen Therapy of Non-Specific Inflammatory Affections, Schweiz, Med. Wchnschr., 73:393-398, 1943.
19. Russ, V. K., Einiges über den Einfluss der Röntgenstrahlen auf Microorganismen, Arch. Hyg., 56:341-360, 1906.
20. Wintz, H., Roentgen Therapy in Inflammatory Diseases, Radiology, 31:156-161, 1938.

WHAT CAN BE DONE FOR THE DEAFENED TODAY*

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CONSIDERABLE publicity has recently been given to the subject of deafness. As a result there has been a deluge of inquiries by both the laity and the profession regarding this subject. This paper is therefore presented to briefly review the etiology, diagnosis and treatment of the various types of deafness.

TYPES OF DEAFNESS

A patient with impaired hearing has one of three types of deafness, namely, conduction or middle ear deafness, perception or nerve deafness, or otosclerosis, which is a combination of both middle ear and nerve involvement. Deafness due to malingering or hysteria is not a true pathological entity, and therefore will not be included in this discussion.

CONDUCTION (MIDDLE EAR) DEAFNESS

Etiology. Any pathology that interferes with the transmission of sound vibrations to the inner ear (cochlea and auditory nerve) results in a conduction deafness. This pathology may be present in the external ear canal, the middle ear, or the eustachian tube. External ear canal obstruction is most commonly due to ceruminosis, furunculosis or foreign bodies. The function of the ear drum may be interfered with by a perforation, edema, or by fibrosis (scars). Sound transmission through the middle ear may be impaired due to ossicular chain pathology, such as dislocation, articular disease, necrosis, or by the presence of fluid, either serous or purulent, in the middle ear. Eustachian tube obstruction results in pressure changes within the middle ear and likewise interferes with sound transmission. Tubal obstruction may be due to altitude changes, inflammatory edema, allergic edema or adenoid hypertrophy.

Diagnosis. The diagnosis of conduction deafness may be made on the basis of subjective findings, objective findings, tuning fork tests and audiograms. The subjective findings in conduction deafness are quite characteristic. The patient usually has a soft-spoken voice, because external noise interference has been diminished, and he therefore hears his own voice very loudly. In conduction deafness, low tones are not heard, and therefore the patient will state he hears better in a noisy environment. This occurs because other people to whom he is talking are conscious of the noisy environment and speak with more intensity. Crunchy foods sound very loud to the conductive deaf individual. The objective findings in conduction deafness may reveal external ear, ear drum or eustachian tube pathology. A 512 tuning fork placed in the center of the forehead of a conductive deaf patient will be referred to the ear with the greater impairment, due to the exclusion of extrinsic sounds (Weber test). A 512 fork no longer heard when held one inch from the external ear will be heard when placed in contact with the mastoid bone (Rinne test). A reading taken with the audiometer will reveal loss of air-borne tones.

Treatment. Treatment of the patient with conduction deafness depends on correction of the underlying pathology or of furnishing adequate substitution, as the individual case requires. Removal of any external ear canal obstruction is imperative. Correction of any ear drum pathology when possible, such as closure of an old perforation, is very desirable. This procedure is successful only in dry, central perforations. It is accomplished by freshening the edges with a caustic and placing scotch tape over the opening, thus allowing epithelization to occur under the paper. Marginal perforations, unfortunately, cannot be closed in this manner. Conduction deafness due to active middle ear disease necessitates our every effort in an attempt to stop further destruction to the hearing mechanism. If drainage persists in acute otitis media for six weeks, in spite of adequate treatment, it is strongly suggestive of bone necrosis in the mastoid cells. If a prophylactic, simple mastoidectomy is not performed at this time, a chronic discharging ear may result. If the acute ear becomes a chronic discharging ear, a permanent conductive deafness results. If the hearing loss is bilateral and is sufficient to produce difficulty in conversation, then a substitute, such as the artificial ear drum or the hearing aid, becomes necessary.

Artificial ear drums (Pohlman inserts) are indicated in cases of bilateral conduction deafness due to chronic ear disease with partial or total destruction of the ear drum and ossicles. A testing probe is used to determine the sensitive contact point on the medial wall of the middle ear. This area is usually about the oval or round window or over the promontory of the cochlea. When this area is contacted with the testing probe, the patient's hearing will suddenly be markedly improved. The insert of choice is then fitted to the ear canal so as to contact this sensitive area. If this area cannot be contacted by the insert, due to drum or ossicular remnants, an ossiculectomy may be necessary. Artificial drums are of several types, such as the cotton pledget, the cellophane cone, moulded rubber (Korogel) and the diaphragm rod. The choice in a given case depends on the tolerance of the individual, whether the ear is moist or dry and on which insert gives the greater decibel boost in the conversational range of hearing (512 to 2048 vibrations per second). Improvement of twenty to thirty decibels is obtained in selected cases by the use of the artificial drum. In these instances, the results obtained are most satisfactory, in that the necessity for an electrical aid is eliminated and the transmission of normal tone sounds is made possible. The patient is taught to make the artificial drum and to remove and replace it as desired.

All persons with conductive deafness can hear with the magnification of sounds made possible by the electrical aid. When other means fail, the electrical aid serves this type of deafness very well. Its disadvantages, such as the cosmetic effect—with the resultant psychological manifestations, the battery nuisance, and the telephone type of transmitted tone, are well known.

Altitude changes and upper respiratory infections frequently result in obstruction of the eustachian tube, due to edema or accumulated mucoid material. If the tube

* Read before the Postgraduate Conference of the Alumni Association of the College of Medical Evangelists.

does not open spontaneously, an inflation or two will usually clear the lumen and restore hearing.

Allergic edema of the tube may result in a prolonged bilateral conduction impairment. In these cases, there is usually an associated allergic rhinitis. The deafness is temporarily relieved by inflation and by the subcutaneous injection of adrenalin, as can be illustrated by audiograms taken before and 20 minutes after its administration. The amount used should be just sufficient to produce an increased cardiac action. After the diagnosis of an allergic tubal obstruction is made, a complete allergic study, followed by the necessary desensitization, is indicated in all cases.

Lymphoid hypertrophy in the nasal pharynx is a frequent cause of conduction deafness in children. Any child with a conduction impairment without evidence of acute ear disease is entitled to a thorough adenoidectomy. Following surgery, adequate radiation therapy should be given to the nasopharyngeal area to further eradicate every vestige of lymphoid tissue. I have seen cases in this classification obtain a 30 decibel boost bilaterally after such treatment.

PERCEPTION (NERVE) DEAFNESS

Etiology. Any pathology which interferes with the function of the cochlea or the pathways of the auditory portion of the eighth nerve results in perception or nerve deafness. These include allergy (edema of the cochlea), infection (labyrinthitis), toxemias, drug (alcohol, quinine, etc.), nervous system disease (multiple sclerosis, syphilis, eighth nerve tumors), hemorrhage, industrial trauma (exposure to a continuous noise level) and skull fractures which involve the temporal bone. Congenital malformation of the auditory pathway is not an uncommon cause of nerve deafness.

Diagnosis. Subjectively the perceptive deaf patient speaks loudly because he does not hear his own voice. Noisy environments markedly interfere with the patient's ability to hear. Objectively the examination may fail to reveal any evidence of ear pathology. A 512 tuning fork placed in the center of the forehead is referred to the ear of lesser impairment. A 512 fork no longer heard when held one inch from the external ear will likewise not be heard when held in contact with the mastoid bone. Readings taken with the audiometer will reveal loss of both air and bone borne tones in approximately the same degree. In contrast to conduction deafness, high tones disappear first in nerve deafness.

Treatment. Generally speaking, the treatment of perceptive deafness is for the most part unsatisfactory. Once the nerve is damaged, it shows little inclination to regenerate, and therefore treatment is directed to prevent further nerve loss. A complete history and a thorough physical examination are indicated in all cases to eliminate every possible underlying cause. The use of massive doses of thiamine chloride has been advocated by many writers. In my experience, the results have been discouraging. The hearing aid is of marked benefit unless the nerve function has been destroyed beyond the point of serviceable hearing. Rehabilitation and the development of the art of lip reading become essential for those with a marked nerve loss. If the loss is congenital, proper speech training beginning in the pre-school age is absolutely imperative.

OTOSCLEROSIS

Etiology. The pathology of clinical otosclerosis consists of a developmental proliferation of bone involving the area about the footplate of the stapes and the oval window. The exact cause of this proliferation is not known, other than it is hereditary in nature.

The incidence of otosclerotic changes in the labyrinthine capsule is very high and is often found at autopsy in persons never known to be deaf. How many persons go through life with otosclerosis which never manifests itself by involving the stapes footplate is not known, but is estimated at 10 per cent. Otosclerosis accounts for 65 per cent of all deafness in this country.

Diagnosis. The diagnosis of otosclerosis may be extremely difficult. Usually otosclerosis is characterized by a progressive bilateral loss of hearing involving first the conductive and later the perceptive mechanism. It becomes apparent in early adult life and is frequently associated with tinnitus. There may or may not be a positive family history of deafness. Frequently the patient has some reason for deafness, such as trauma, childhood disease, or a recent upper respiratory infection. This, unfortunately, may cause the examining doctor to err in the diagnosis of otosclerosis.

A very thorough examination of the entire ear mechanism will fail to elicit any cause for the patient's impairment. The eustachian tubes inflate readily, the ear drums appear thin and translucent, and no apparent pathology can be noted. Interestingly enough, one seldom finds cerumen in the external ear of a patient with otosclerosis.

By the time the patient seeks medical aid, an audiogram will reveal a bilateral conduction loss of twenty-five to fifty decibels, with or without evidence of nerve involvement.

Any progressive bilateral hearing impairment occurring in early or middle adult life, which on examination does not present evidence of ear pathology, very strongly suggests otosclerosis. This coupled with a co-existant tinnitus and a family history of deafness is almost diagnostic of otosclerosis.

Treatment. Perhaps no other group of individuals has been subjected to more treatment without benefit than has the unfortunate patient with otosclerosis. Repeated eustachian tube inflations, nose treatments, nose and throat surgery, ear drum massage, various diets, endocrines, vitamins and minerals all have been put through their paces without noticeable benefit. When benefit has been derived, one is led to believe the diagnosis of otosclerosis was in error, for it is difficult when one visualizes the underlying pathology to understand how such therapy could have much reward.

Some seventy years ago an otologist by the name of Kessel had a patient who was suffering from otosclerosis. The patient subsequently heard normally in one ear following a skull fracture involving the temporal bone. Years later an autopsy revealed an open crack in the bone over the patient's intact membranous labyrinth.

This led to considerable experimental work in creating surgically an opening in the osseous labyrinth. It was noted all otosclerotics heard well as soon as this opening was made, providing their nerve function was capable of transmitting the sound to the brain. Likewise, as soon as the bony opening closed, their hearing dropped back to its original level.

Passow, Jenkins, Barany, Holmgren and Sourdille are prominent names in the history of otosclerotic surgery and each made his contribution.

Some ten years ago Doctor Julius Lempert** developed the first satisfactory surgical technique which could be employed routinely in these cases.* Since then he has modified the procedure on several occasions until now the bony window made over the labyrinth remains permanently patent in some 65 per cent of the cases operated. If the opening closes, it will usually do so within the

* Ed. Note. For special article on "Fenestration Operation for Deafness," by Robert C. Martin, M.D., see CALIFORNIA AND WESTERN MEDICINE, June, 1945, page 311.

** Fenestra Nov-Ovalis, Julius Lempert, Archives of Otolaryngology, Jan. 1945, Vol. 41, pp. 1-41.

first six months after surgery. Oddly enough, if the opening closes once, the chances are it will close again if reopened. However, because the opening has closed in one ear is no indication it will close if the other ear is operated. We have no way of knowing beforehand which cases are going to close and which will remain open. Considerable research is being conducted at the present time in an attempt to discover some means of keeping the bony opening patent in all cases. Gold burnishing, saucer-shaping the fenestra, metal and cartilage inserts, etc., have all been used, and none has proven infallible.

As one may well realize, surgery of this type is very delicate and exacting. Fortunately the incidence of serious complications is less than 2 per cent. These complications include labyrinthine damage from injury or infection, facial paralysis, meningitis, etc. There have been no deaths directly attributable to the operation up to the present time.

It has been noted the nerve function of the successfully operated otosclerotic seems to remain at its preoperative level instead of gradually deteriorating, as in the unoperated case. This has not been satisfactorily explained, for it is obvious the surgery in no way influences the disease of otosclerosis but merely short-circuits the involved area so far as the hearing mechanism is concerned. It is thought the progressive nerve loss in the unoperated otosclerosis may be on the basis of a disuse atrophy.

Today the patient with otosclerosis has but one of two choices when the hearing loss becomes pronounced. One is to turn to the hearing aid and the other is the fenestration operation. The hearing aid, within its limitations, will give very satisfactory results in early otosclerosis. Ultimately, if the patient lives long enough and if the otosclerotic process advances to involve the nerve beyond the conversational range, even the hearing aid is of little or no value, and the patient must turn to rehabilitation and lip-reading, just as in the case of far-advanced nerve deafness.

The fenestration operation is indicated in any case of otosclerosis in which the ear drum is intact and the nerve function is capable of transmitting the normal ranges of conversation to the brain. The degree of nerve function present is determined by tuning fork tests and audiometric readings. If the patient has these qualifications, the fenestration operations offers a 60 per cent chance of having serviceable hearing permanently restored. Barring complications, if the fenestra should close postoperatively, the patient's hearing will drop to its previous level. In this event, either the same ear can be reoperated, the opposite ear can be operated, or the patient may turn to the hearing aid and hope his eighth nerve outlives his need for its function.

It is the hope of all of us performing this surgery that some means will be found to keep the newly-created fenestra open in all cases. When this is accomplished, the patient with otosclerosis will have little cause to fear the future.

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Charles Dickens (1812-1870).—The sickly, sensitive child was father to the restless, overwrought man. Exhaustion, insomnia, failing sight and memory, these were the physical infirmities that burdened Dickens during the latter years of his productive career. In 1864, he became afflicted with a lameness that grew steadily worse. In 1865, he was in a railway accident. Though able to aid those more badly injured than he, he suffered from the shock incurred. In "Edwin Drood" there are unmistakable signs of his dimming faculties.—Warner's *Calendar of Medical History.*

CLINICAL NOTES AND CASE REPORTS

SULFAPYRIDINE AS A HEMOSTATIC AGENT

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It is not generally known that powdered sulfapyridine applied locally is a very effective hemostatic agent. In 1942 Cunningham[1] reported on its use. He found that the instillation of sulfamethylthiazole powder in the operative wound after radical external frontal sinusotomy for frontal bone osteomyelitis prevented the usual considerable postoperative oozing. This observation stmiulated him to experiment on guinea-pigs. He made circular wounds on the backs of the animals and five to seven days later, he evulsed the scabs so that an open granulating and freely oozing surface remained. Using a powder blower he sprayed powdered sulfapyridine, sulfanilamide, sulfathiazole, sulfamethylthiazole and talc on the wounds. The wounds treated with sulfamethylthiazole and sulfapyridine stopped oozing at once while the wounds treated with the other powders and the control wounds, all continued to ooze for from five to ten minutes. The sulfapyridine and sulfamethylthiazole powders also proved to be bacteriostatic. These results led Cunningham to employ powdered sulfapyridine and sulfamethylthiazole by insufflation in the control of postoperative secondary tonsillar hemorrhage with excellent results.

Stimulated by Cunningham's report, I have used it extensively in a dermatologic practice. Occasionally after electrodesiccation or electrocoagulation of a cutaneous lesion, separation of the eschar will be followed by an annoying persistent bleeding. This is especially so after removal of a carcinoma or keratosis of the lower lip where the frequent movements of the lip tend to prematurely force the separation of the eschar. The application of sulfapyridine powder will instantly control this bleeding. I now routinely apply sulfapyridine powder to all wounds after removal of cutaneous lesions by electrosurgery or cautery. The surfaces of the wounds become hard and dry and heal without infection. The sulfapyridine powder is also very useful in the treatment of bleeding abrasions of the skin.

The use of sulfapyridine powder as a hemostatic agent can be extended into other specialties besides those of dermatology and otorhinolaryngology. The dentist and the general practitioner should find it extremely useful.

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REFERENCES

1. Cunningham, B. P.: Clinical and Experimental Studies with Sulfapyridine as a Hemostatic Agent, Collected Papers of the Mayo Clinic, 39:12, 1942, Philadelphia, W. B. Saunders Company, 1943.

Voltaire (1604-1778).—Born of a tuberculous mother, the great forerunner of the French Revolution, at one time imprisoned in the Bastille, complained throughout his life of a formidable list of maladies which he sometimes used as a shield against his enemies. Excessively thin from childhod, he became a mere skeleton in old age. Yet he was possessed of untiring energy and an extraordinary capacity for work. Poet, dramatist, philosopher, Voltaire remains a commanding figure in literature. When he died only three words were needed on his tombstone: *"Ici reste Voltaire."*—Warner's *Calendar of Medical History.*